



BILLING CODE 6560-50-P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[EPA-HQ-OPP-2012-0588; FRL-9909-72]

Fenoxaprop-ethyl; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a tolerance for residues of fenoxaprop-ethyl (FE), in or on grass hay. Interregional Research Project Number 4 (IR-4) requested this tolerance under the Federal Food, Drug, and Cosmetic Act (FFDCA).

DATES: This regulation is effective [*insert date of publication in the Federal Register*].

Objections and requests for hearings must be received on or before [*insert date 60 days after date of publication in the Federal Register*], and must be filed in accordance with the instructions provided in 40 CFR part 178 (see also Unit I.C. of the

SUPPLEMENTARY INFORMATION).

ADDRESSES: The docket for this action, identified by docket identification (ID) number EPA-HQ-OPP-2012-0588, is available at <http://www.regulations.gov> or at the Office of Pesticide Programs Regulatory Public Docket (OPP Docket) in the Environmental Protection Agency Docket Center (EPA/DC), EPA West Bldg., Rm. 3334, 1301 Constitution Ave., NW., Washington, DC 20460-0001. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566-1744, and the

telephone number for the OPP Docket is (703) 305-5805. Please review the visitor instructions and additional information about the docket available at

<http://www.epa.gov/dockets>.

FOR FURTHER INFORMATION CONTACT: Lois Rossi, Registration Division (7505P), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460-0001; telephone number: (703) 305-7090; email address: *RDFRNotices@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. The following list of North American Industrial Classification System (NAICS) codes is not intended to be exhaustive, but rather provides a guide to help readers determine whether this document applies to them. Potentially affected entities may include:

- Crop production (NAICS code 111).
- Animal production (NAICS code 112).
- Food manufacturing (NAICS code 311).
- Pesticide manufacturing (NAICS code 32532).

B. How Can I Get Electronic Access to Other Related Information?

You may access a frequently updated electronic version of EPA's tolerance regulations at 40 CFR part 180 through the Government Printing Office's e-CFR site at *http://www.ecfr.gov/cgi-bin/text-idx?&c=ecfr&tpl=/ecfrbrowse/Title40/40tab_02.tpl*.

C. How Can I File an Objection or Hearing Request?

Under FFDCFA section 408(g), 21 U.S.C. 346a, any person may file an objection to any aspect of this regulation and may also request a hearing on those objections. You must file your objection or request a hearing on this regulation in accordance with the instructions provided in 40 CFR part 178. To ensure proper receipt by EPA, you must identify docket ID number EPA-HQ-OPP-2012-0588 in the subject line on the first page of your submission. All objections and requests for a hearing must be in writing, and must be received by the Hearing Clerk on or before *[insert date 60 days after date of publication in the **Federal Register**]*. Addresses for mail and hand delivery of objections and hearing requests are provided in 40 CFR 178.25(b).

In addition to filing an objection or hearing request with the Hearing Clerk as described in 40 CFR part 178, please submit a copy of the filing (excluding any Confidential Business Information (CBI)) for inclusion in the public docket. Information not marked confidential pursuant to 40 CFR part 2 may be disclosed publicly by EPA without prior notice. Submit the non-CBI copy of your objection or hearing request, identified by docket ID number EPA-HQ-OPP-2012-0588, by one of the following methods:

- *Federal eRulemaking Portal*: <http://www.regulations.gov>. Follow the online instructions for submitting comments. Do not submit electronically any information you consider to be CBI or other information whose disclosure is restricted by statute.
- *Mail*: OPP Docket, Environmental Protection Agency Docket Center (EPA/DC), (28221T), 1200 Pennsylvania Ave., NW., Washington, DC 20460-0001.

• *Hand Delivery*: To make special arrangements for hand delivery or delivery of boxed information, please follow the instructions at

<http://www.epa.gov/dockets/contacts.html>.

Additional instructions on commenting or visiting the docket, along with more information about dockets generally, is available at <http://www.epa.gov/dockets>.

II. Summary of Petitioned-For Tolerance

In the **Federal Register** of September 28, 2012 (77 FR 59578) (FRL-9364-6), EPA issued a document pursuant to FFDCA section 408(d)(3), 21 U.S.C. 346a(d)(3), announcing the filing of a pesticide petition (PP 2E8051) by IR-4, 500 College Road East, Suite 201W., Princeton, NJ 08540. The petition requested that 40 CFR 180.430 be amended by establishing tolerances for residues of the herbicide fenoxaprop-ethyl, [(±)-ethyl 2-[4- [(6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoate] and its metabolites 2-[4- [(6-chloro-2-benzoxazolyl) oxy]phenoxy] propanoic acid and 6-chloro-2,3-dihydrobenzoxazol-2-one, each expressed as the parent compound, in or on grass, hay at 0.15 part per million (ppm). Based on the regional residue data submitted from Washington and Oregon, and the petitioner's intent for this to be a regional pesticide tolerance, the tolerance is being established as a "Tolerance with regional registration" with use restricted to Oregon, Washington, and Utah. That document referenced a summary of the petition prepared by Bayer CropScience, the registrant, which is available in the docket, <http://www.regulations.gov>.

Comments were received on the notice of filing. EPA response to those comments is discussed in Unit IV.C.

Based upon review of the data supporting the petition, EPA has modified the level at which the tolerance is being established. The reason for this change is explained in Unit IV.D.

III. Aggregate Risk Assessment and Determination of Safety

Section 408(b)(2)(A)(i) of FFDCA allows EPA to establish a tolerance (the legal limit for a pesticide chemical residue in or on a food) only if EPA determines that the tolerance is “safe.” Section 408(b)(2)(A)(ii) of FFDCA defines “safe” to mean that “there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information.” This includes exposure through drinking water and in residential settings, but does not include occupational exposure. Section 408(b)(2)(C) of FFDCA requires EPA to give special consideration to exposure of infants and children to the pesticide chemical residue in establishing a tolerance and to “ensure that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to the pesticide chemical residue....”

Consistent with FFDCA section 408(b)(2)(D), and the factors specified in FFDCA section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action. EPA has sufficient data to assess the hazards of and to make a determination on aggregate exposure for fenoxaprop-p-ethyl (FPE) including exposure resulting from the tolerances established by this action. EPA's assessment of exposures and risks associated with FPE follows.

A. Toxicological Profile

EPA has evaluated the available toxicity data and considered its validity, completeness, and reliability as well as the relationship of the results of the studies to human risk. EPA has also considered available information concerning the variability of the sensitivities of major identifiable subgroups of consumers, including infants and children.

FPE is an enriched isomer formulation (95% *d* and 5% *l* enantiomers) based on the previously registered product FE which is a 50:50 racemic mixture of *d* and *l* enantiomers. FE is no longer a registered active ingredient. The toxicology database for FPE is complete based on studies submitted for both FPE and FE. Based on the analysis of the submitted studies, EPA found that the toxicological effects of FE and FPE across species, duration, and route of exposure are similar. Most of the toxicological data available involved testing of the FE, not FPE. However, EPA has concluded that the similarity between the FE and FPE data is such that the database for FE could be bridged with FPE.

The major target organs following short-term and long-term oral administration of FE and FPE in rats and mice are the liver and kidneys, with rats being the most sensitive species. The primary toxic effect is altered lipid metabolism characterized by decreased lipids and cholesterol, and increased liver weights in rats, and slightly increased lipids, cholesterol, proteins, and liver weights in mice. Additionally, increased enzyme activity (aspartate amino transferase (ASAT), alanine amino transferase (ALAT), and alkaline phosphatase (ALP)), hypertrophy, and single cell necrosis were observed in mice. In the kidneys, increases in ketones and kidney weights were observed in rats and evidence of proximal renal tubular injury were observed in mice following 90-day administration of

FPE. However, no effects on the kidneys were observed following chronic administration of FE to rats, mice, or dogs. In both species, males were slightly more sensitive to the liver effects of FE and FPE. It is also important to note that no increases in toxicity are observed over time for FE when comparing the 28-day and 90-day subchronic studies, the 2-generation reproductive toxicity study, and the 2-year chronic toxicity/carcinogenicity study in rats, or in FPE when comparing the 28-day and 90-day subchronic toxicity studies in rats.

FPE has low acute toxicity following the oral, dermal, and inhalation routes of exposure. No evidence of immunotoxicity, reproductive or neurological toxicity was identified in the database. Developmental toxicity occurred in rats as evidenced by skeletal anomalies (longitudinally displaced, fragmented, fused, dysplastic sternebrae or dislocated sternebrae) and skeletal retardations (weak or non-ossification of one or several cranial bones). Developmental effects only occurred in the rat in the presence of maternal toxicity (decreased body weight, body weight gain, and heart weight). No developmental effects were identified in rabbits. In mice, a treatment-related increase in tumor incidence of hepatocellular adenomas and carcinomas, mainly adenomas, was observed in males at 320 ppm (30%) compared to the control (2%). In addition, microscopic pathology indicated that hepatocellular hypertrophy was observed in the majority of treated animals (both sexes). There was, however, no evidence of a mutagenic effect in a comprehensive battery of genetic toxicology assays with both isomers. No evidence of tumors was identified in rats.

The only tumor response induced by FE/FPE occurred in the liver of male mice; no liver tumors were seen in the female mice or in the guideline chronic/carcinogenicity

study in male and female rats. The tumors were benign with no progression to malignancy. Mutagenicity has been ruled out as a mode of action (MOA) for this response. The presence of a single non- mutagenic tumor type in one sex and species – here, benign liver tumors in the male mouse, a common tumor in mice – provides no more than a weak suggestion of possible carcinogenic effects and thus does not support a linear assessment of risk based on the tumor incidence. Given the doses at which the benign mouse tumors were seen, EPA concludes that the chronic reference dose (cRfD) for FPE will adequately protect for all chronic toxicity, including carcinogenicity, that could result from exposure to FE/FPE.

The Agency has waived the requirements for acute and subchronic neurotoxicity studies based on the following rationale:

1. The lack of neurotoxicity in the available toxicology database for FE and FPE.
2. The target organs of FPE and FE are the kidney and liver, and the mechanism of action for FPE and the chemical class do not target the nervous system.
3. Developmental effects and decreased total blood lipids/cholesterol are the most sensitive effects seen in the FE database and provide the most sensitive POD for risk assessment.
4. There is low concern for neurotoxicity in other members of this class of chemicals (*i.e.*, the arloxy phenoxy-propionate class).

Specific information on the studies received and the nature of the adverse effects caused by FPE as well as the no-observed-adverse-effect-level (NOAEL) and the lowest-observed-adverse-effect-level (LOAEL) from the toxicity studies can be found at <http://www.regulations.gov> in the document titled “*Fenoxaprop-p-ethyl. Registration*

Review Preliminary Risk Assessment and Proposed New Use on Grass Grown for Seed’ on pages 52-57 in docket ID number EPA-HQ-OPP-2012-0588.

B. Toxicological Points of Departure/Levels of Concern

Once a pesticide’s toxicological profile is determined, EPA identifies toxicological points of departure (POD) and levels of concern (LOC) to use in evaluating the risk posed by human exposure to the pesticide. For hazards that have a threshold below which there is no appreciable risk, the toxicological POD is used as the basis for derivation of reference values for risk assessment. PODs are developed based on a careful analysis of the doses in each toxicological study to determine the dose at which no adverse effects are observed (the NOAEL) and the lowest dose at which adverse effects of concern are identified (the LOAEL). Uncertainty/safety factors are used in conjunction with the POD to calculate a safe exposure level - generally referred to as a population-adjusted dose (PAD) or a reference dose (RfD) - and a safe margin of exposure (MOE). For non-threshold risks, the Agency assumes that any amount of exposure will lead to some degree of risk. Thus, the Agency estimates risk in terms of the probability of an occurrence of the adverse effect expected in a lifetime. For more information on the general principles EPA uses in risk characterization and a complete description of the risk assessment process, see

<http://www.epa.gov/pesticides/factsheets/riskassess.htm>.

A summary of the toxicological endpoints for FPE used for human risk assessment is shown in Table 1 of this unit.

Table 1--Summary of Toxicological Doses and Endpoints for FPE for Use in Human Health Risk Assessment

Exposure/Scenario	POD and Uncertainty/Safety Factors	RfD, PAD, LOC for Risk Assessment	Study and Toxicological Effects
Acute dietary (general population including infants and children and females 13-50 years of age)	No appropriate endpoint attributable to a single dose was identified. An acute RfD was not established.		
Chronic dietary (all populations)	NOAEL = 1.5 mg/kg/day UF _A = 10x UF _H = 10x FQPA SF = 1x	Chronic RfD = 0.015 mg/kg/day cPAD = 0.015 mg/kg/day	Chronic toxicity/carcinogenicity (rat) 2-generation reproductive toxicity (rat) LOAEL = 9 mg/kg/day, based on decreased serum lipids and cholesterol, and altered liver weights
Incidental oral short-term (1 to 30 days)	NOAEL = 6 mg/kg/day UF _A = 10x UF _H = 10x FQPA SF = 1x	LOC for MOE = 100	28-day oral toxicity (rat) LOAEL = 26 mg/kg/day, based on altered lipid metabolism (decreased HDL-cholesterol, HDL-phospholipids, and total lipids, increased triglycerides, and ketonuria) and increased liver and kidney weights.
Dermal short-term (1 to 30 days) and intermediate-term (1 to 6 months)	Dermal study NOAEL = 20 mg/kg/day UF _A = 10x UF _H = 10x FQPA SF = 1x	LOC for MOE = 100	28-day dermal toxicity (rat) LOAEL = 100 mg/kg/day, based on non-regenerative anemia, decreased serum cholesterol, total lipids, and protein (beta 1 globulins), and increased liver and kidney weights were observed. Additionally, cholesterol remained decreased following a 15-day recovery period
Inhalation short-term (1-30 days) and intermediate-term (1 – 6 months)	Inhalation study NOAEL = 0.07 mg/L (males) 0.3 mg/L (females) UF _A = 3x UF _H = 10x FQPA SF = 1x	LOC for MOE = 30	21-day inhalation toxicity (rat) LOAEL = 0.3 mg/L (males only) Based on slight normocytic anemia, decreases in serum cholesterol and total lipids, and increases in liver weight and urea nitrogen.

Cancer (oral, dermal, inhalation)	Quantification of risk using a non-linear approach; <i>i.e.</i> , RfD, for FPE will adequately account for all chronic toxicity, including carcinogenicity, that could result from exposure to FPE
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FQPA SF = Food Quality Protection Act Safety Factor. LOAEL = lowest-observed-adverse-effect-level. LOC = level of concern. mg/kg/day = milligram/kilogram/day. MOE = margin of exposure. NOAEL = no-observed-adverse-effect-level. PAD = population adjusted dose (a = acute, c = chronic). RfD = reference dose. UF = uncertainty factor. UF_A = extrapolation from animal to human (interspecies). UF_H = potential variation in sensitivity among members of the human population (intraspecies).

C. Exposure Assessment

1. *Dietary exposure from food and feed uses.* In evaluating dietary exposure to FPE, EPA considered exposure under the petitioned-for tolerances as well as all existing FE tolerances in 40 CFR 180.430. EPA assessed dietary exposures from FPE and FE in food as follows:

i. *Acute exposure.* Quantitative acute dietary exposure and risk assessments are performed for a food-use pesticide, if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a 1-day or single exposure.

No such effects were identified in the toxicological studies for FPE; therefore, a quantitative acute dietary exposure assessment is unnecessary.

ii. *Chronic exposure.* In conducting the chronic dietary exposure assessment EPA used the food consumption data from the United States Department of Agriculture's (USDA's) National Health and Nutrition Examination Survey, What We Eat in America, (NHANES/WWEIA). As to residue levels in food, EPA use an unrefined analysis based on tolerance-level residues, 100 percent crop treated (PCT) assumptions, and Dietary Exposure Evaluation Model (DEEM) default processing factors.

iii. *Cancer.* Based on the data summarized in Unit III.A., EPA has concluded that a nonlinear RfD approach is appropriate for assessing cancer risk to FPE. Cancer risk was assessed using the same exposure estimates as discussed in Unit III.C.1.ii.

iv. *Anticipated residue and PCT information.* EPA did not use anticipated residue or PCT information in the dietary assessment for FPE or FE. Tolerance level residues and 100 PCT were assumed for all food commodities.

2. *Dietary exposure from drinking water.* The Agency has identified FPE and its three degradates, fenoxaprop-p acid ((D+)-2-[4-(6-chloro-2-benzoxazolyloxy) phenoxy] propanoate, AE F088406), chlorobenzoxazolone (4-(6-chloro-2-benzoxazolyloxy) phenol, AE F054014), and 4-(6-chloro-2-benzoxazolyloxy) phenol (AE F040356), as residues of concern in drinking water. The parent plus the three degradates were assessed using a total toxic residue (TTR) approach.

The Agency used screening level water exposure models in the dietary exposure analysis and risk assessment for FPE and its three degradates in drinking water. These simulation models take into account data on the physical, chemical, and fate/transport characteristics of FPE and its three degradates. Further information regarding EPA drinking water models used in pesticide exposure assessment can be found at <http://www.epa.gov/oppefed1/models/water/index.htm>.

Based on the Tier 1 Rice Model and Pesticide Root Zone Model Ground Water (PRZM GW), the estimated drinking water concentrations (EDWCs) of FPE and its degradates (TTR) for chronic exposure assessments are estimated to be 68.6 parts per billion (ppb) for surface water and 0.032 ppb for ground water.

Modeled estimates of drinking water concentrations were directly entered into the dietary exposure model. For chronic dietary risk assessment, the water concentration of value 68.6 ppb was used to assess the contribution to drinking water.

3. *From non-dietary exposure.* The term “residential exposure” is used in this document to refer to non-occupational, non-dietary exposure (e.g., for lawn and garden pest control, indoor pest control, termiticides, and flea and tick control on pets). FPE is currently registered for the following uses that could result in residential exposures: Residential turf and home garden. EPA assessed residential exposure using the following assumptions: For residential handlers, both short-term dermal and short-term inhalation exposure is expected as a result of applying FPE to ornamentals and turf.

There is the potential for short-term dermal and incidental short-term oral post-application exposure for individuals exposed as a result of being in an environment that has been previously treated with FPE. The quantitative exposure/risk assessment for residential post-application exposures is based on the following scenarios:

- ☐rAdults High Contact Lawn Activities
- ☐dChildren 1 to < 2 years old High Contact Lawn Activities
- ☐ Adults Mowing Turf
- ☐dChildren 11 to < 16 years old Mowing Turf
- ☐hAdults Ornamental Garden Activities
- ☐dChildren 6 to < 11 years old Ornamental Garden Activities

The most conservative residential exposure scenario for adults reflects dermal exposure from post-application exposure to turf and gardens. The most conservative

residential exposure for children reflects dermal and hand-to-mouth exposures from post-application high contact lawn activity exposure from turf applications.

Further information regarding EPA standard assumptions and generic inputs for residential exposures may be found at

<http://www.epa.gov/pesticides/trac/science/trac6a05.pdf>.

4. *Cumulative effects from substances with a common mechanism of toxicity.*

Section 408(b)(2)(D)(v) of FFDCA requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider “available information” concerning the cumulative effects of a particular pesticide's residues and “other substances that have a common mechanism of toxicity.”

EPA has not found FPE to share a common mechanism of toxicity with any other substances, and FPE does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has assumed that FPE does not have a common mechanism of toxicity with other substances. For information regarding EPA's efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such chemicals, see EPA's website at <http://www.epa.gov/pesticides/cumulative>.

D. *Safety Factor for Infants and Children*

1. *In general.* Section 408(b)(2)(C) of FFDCA provides that EPA shall apply an additional tenfold (10X) margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the database on toxicity and exposure unless EPA determines based on reliable data that a different margin of safety will be safe for infants and children. This additional margin of safety is

commonly referred to as the FQPA Safety Factor (SF). In applying this provision, EPA either retains the default value of 10X, or uses a different additional safety factor when reliable data available to EPA support the choice of a different factor.

2. *Prenatal and postnatal sensitivity.* The available data do not provide evidence of any increased susceptibility in the offspring in either of the two developmental toxicity studies for FPE or in the 2-generation reproduction study for FE. Delayed ossification was the primary effect in the developmental toxicity study and only occurred in the presence of maternal toxicity and a clearly defined NOAEL and LOAEL were achieved.

In the rat developmental toxicity study with FPE, longitudinally displaced, fragmented, fused, dysplastic sternebrae or dislocated sternebrae and weak or non-ossification of one or several cranial bones were noted at 100 mg/kg (highest dose tested). These incidences occurred only in the presence of maternal toxicity (decreased gestational body weights, body weight gains, and food consumption). No developmental effects occurred in rabbits. In the 2-generation rat reproductive toxicity study on FE, no reproductive or developmental effects were observed. An increase in ALP activity and liver weights were identified in the offspring at 9.0 mg/kg. These effects occurred in the presence of parental toxicity (increased liver weight and decreased lipids) and are consistent with hepatotoxicity, the primary toxic effect of FPE, observed across the database.

3. *Conclusion.* EPA has determined that reliable data show the safety of infants and children would be adequately protected if the FQPA SF were reduced to 1x. That decision is based on the following findings:

- i. The toxicity database for FPE is complete.

ii. There is no indication that FPE is a neurotoxic chemical and there is no need for a developmental neurotoxicity study or additional UFs to account for neurotoxicity.

iii. There is no evidence that FPE results in increased susceptibility in *in utero* rats or rabbits in the prenatal developmental studies or in young rats in the 2-generation reproduction study.

iv. There are no residual uncertainties identified in the exposure databases. The dietary food exposure assessments were performed based on 100 PCT and tolerance-level residues. EPA made conservative (protective) assumptions in the ground and surface water modeling used to assess exposure to FPE in drinking water. EPA used similarly conservative assumptions to assess post-application exposure of children as well as incidental oral exposure of toddlers. These assessments will not underestimate the exposure and risks posed by FPE.

E. Aggregate Risks and Determination of Safety

EPA determines whether acute and chronic dietary pesticide exposures are safe by comparing aggregate exposure estimates to the acute PAD (aPAD) and chronic PAD (cPAD). For linear cancer risks, EPA calculates the lifetime probability of acquiring cancer given the estimated aggregate exposure. Short-, intermediate-, and chronic-term risks are evaluated by comparing the estimated aggregate food, water, and residential exposure to the appropriate PODs to ensure that an adequate MOE exists.

1. *Acute risk.* An acute aggregate risk assessment takes into account acute exposure estimates from dietary consumption of food and drinking water. No adverse effect resulting from a single oral exposure was identified and no acute dietary endpoint was selected. Therefore, FPE is not expected to pose an acute risk.

2. *Chronic risk.* Using the exposure assumptions described in this unit for chronic exposure, EPA has concluded that chronic exposure to FPE and FE from food and water will utilize 28% of the cPAD for all infants less than 1 year old, the population group receiving the greatest exposure. Based on the explanation in Unit III.C.3., regarding residential use patterns, chronic residential exposure to residues of FPE is not expected.

3. *Short-term risk.* Short-term aggregate exposure takes into account short-term residential exposure plus chronic exposure to food and water (considered to be a background exposure level).

FPE is currently registered for uses that could result in short-term residential exposure, and the Agency has determined that it is appropriate to aggregate chronic exposure through food and water with short-term residential exposures to FPE.

Using the exposure assumptions described in this unit for short-term exposures, EPA has concluded the combined short-term food, water, and residential exposures result in aggregate worst case MOEs of 249 for adults and 302 for children. Because EPA's level of concern for FPE is a MOE of 100 or below, these MOEs are not of concern.

4. *Intermediate-term risk.* Intermediate-term aggregate exposure takes into account intermediate-term residential exposure plus chronic exposure to food and water (considered to be a background exposure level).

An intermediate-term adverse effect was identified; however, FPE is not registered for any use patterns that would result in intermediate-term residential exposure. Intermediate-term risk is assessed based on intermediate-term residential exposure plus chronic dietary exposure. Because there is no intermediate-term residential exposure and

chronic dietary exposure has already been assessed under the appropriately protective cPAD (which is at least as protective as the POD used to assess intermediate-term risk), no further assessment of intermediate-term risk is necessary, and EPA relies on the chronic dietary risk assessment for evaluating intermediate-term risk for FPE.

5. *Aggregate cancer risk for U.S. population.* EPA considers the chronic aggregate risk assessment to be protective of any aggregate cancer risk.

6. *Determination of safety.* Based on these risk assessments, EPA concludes that there is a reasonable certainty that no harm will result to the general population or to infants and children from aggregate exposure to FPE and FE residues.

IV. Other Considerations

A. Analytical Enforcement Methodology

Adequate enforcement methodology (gas chromatography with electron capture detection (GD-ECD) method, based on Hoechst HRAV Analytical Method HRAV-4B) is available to enforce the tolerance expression.

B. International Residue Limits

In making its tolerance decisions, EPA seeks to harmonize U.S. tolerances with international standards whenever possible, consistent with U.S. food safety standards and agricultural practices. EPA considers the international maximum residue limits (MRLs) established by the Codex Alimentarius Commission (Codex), as required by FFDCA section 408(b)(4). The Codex Alimentarius is a joint United Nations Food and Agriculture Organization/World Health Organization food standards program, and it is recognized as an international food safety standards-setting organization in trade agreements to which the United States is a party. EPA may establish a tolerance that is

different from a Codex MRL; however, FFDCA section 408(b)(4) requires that EPA explain the reasons for departing from the Codex level.

The Codex has not established a MRL for FPE in or on grass hay.

C. Response to Comments

Two comments that were received were not related to FPE and therefore, do not need to be addressed here. A third comment was received stating that FPE is an endocrine disruptor and America does not need any more of those. In the available toxicity studies on FPE, there was no estrogen, androgen, and/or thyroid mediated toxicity. The Agency currently has no evidence that FPE is an endocrine disruptor.

D. Revisions to Petitioned-For Tolerances

EPA has modified the tolerance from the proposed level of 0.15 ppm to 0.09 ppm for the following reason: The method used for data-collection (as well as tolerance enforcement) converts the residues of concern for FPE to acyl 6-chlorobenzoxazolone for detection. It is necessary to then convert this residue value to parent equivalents. Since the residues found on grass, hay were less than the limit of quantitation (LOQ) of 0.05 ppm for acyl 6-chlorobenzoxazolone, EPA multiplied this 0.05 ppm value by the ratio of the molecular weights (1.71) to arrive at a recommended tolerance of 0.09 ppm.

V. Conclusion

Therefore, a tolerance with regional registration is established for residues of fenoxaprop-ethyl, [(±)-ethyl 2-[4- [(6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoate] and its metabolites 2-[4-[(6:-chloro-2-benzoxazolyl) oxy]phenoxy] propanoic acid and 6-chloro-2,3-dihydrobenzoxazol-2-one, each expressed as the parent compound, in or on grass, hay at 0.09 ppm.

VI. Statutory and Executive Order Reviews

This final rule establishes tolerances under FFDCA section 408(d) in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled “Regulatory Planning and Review” (58 FR 51735, October 4, 1993). Because this final rule has been exempted from review under Executive Order 12866, this final rule is not subject to Executive Order 13211, entitled “Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use” (66 FR 28355, May 22, 2001) or Executive Order 13045, entitled “Protection of Children from Environmental Health Risks and Safety Risks” (62 FR 19885, April 23, 1997). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA) (44 U.S.C. 3501 *et seq.*), nor does it require any special considerations under Executive Order 12898, entitled “Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations” (59 FR 7629, February 16, 1994).

Since tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerance in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*), do not apply.

This final rule directly regulates growers, food processors, food handlers, and food retailers, not States or tribes, nor does this action alter the relationships or distribution of power and responsibilities established by Congress in the preemption provisions of FFDCA section 408(n)(4). As such, the Agency has determined that this action will not have a substantial direct effect on States or tribal governments, on the

relationship between the national government and the States or tribal governments, or on the distribution of power and responsibilities among the various levels of government or between the Federal Government and Indian Tribes. Thus, the Agency has determined that Executive Order 13132, entitled “Federalism” (64 FR 43255, August 10, 1999) and Executive Order 13175, entitled “Consultation and Coordination with Indian Tribal Governments” (65 FR 67249, November 9, 2000) do not apply to this final rule. In addition, this final rule does not impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (2 U.S.C. 1501 *et seq.*).

This action does not involve any technical standards that would require Agency consideration of voluntary consensus standards pursuant to section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA) (15 U.S.C. 272 note).

VII. Congressional Review Act

Pursuant to the Congressional Review Act (5 U.S.C. 801 *et seq.*), EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of the rule in the **Federal Register**. This action is not a “major rule” as defined by 5 U.S.C. 804(2).

List of Subjects in 40 CFR Part 180

Environmental protection, Administrative practice and procedure, Agricultural commodities, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: April 29, 2014.

Lois Rossi,

Director, Registration Division, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180--[AMENDED]

1. The authority citation for part 180 continues to read as follows:

Authority: 21 U.S.C. 321(q), 346a and 371.

2. In § 180.430, revise paragraph (c) to read as follows:

§ 180.430 Fenoxaprop-ethyl; tolerances for residues.

* * * * *

(c) *Tolerances with regional registrations.* Tolerances with regional registration, as defined in §180.1(l), are established for residues of the herbicide fenoxaprop-ethyl, including its metabolites and degradates, in or on the commodities in the table in this paragraph when fenoxaprop-ethyl is used in the states of Oregon, Washington, and Utah. Compliance with the tolerance levels specified in this paragraph is to be determined by measuring only the sum of fenoxaprop-ethyl, (±)-ethyl 2-[4-[(6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoate, and its metabolites, 2-[4-[(6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoic acid and 6-chloro-2,3-dihydrobenzoxazol-2-one, calculated as the stoichiometric equivalent of fenoxaprop-ethyl, in or on the commodity

Commodity	Parts per million
Grass, hay	0.09

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